ORIGINAL ARTICLE

CONTENTS

Advances in clinical and metabolic profiling in children with type 1 diabetes and diabetic nephropathy

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ABSTRACT

Aim: To stratify children with T1D according to their risk of developing and progressing DN using cluster analysis of clinical and laboratory indicators.

Materials and Methods: Cluster analysis was performed in a cohort of 60 children (mean age 13 ± 3 years; 17 [28,3%] boys and 43 [71,7%] girls) with T1D, with (n = 30) or without (n = 30) early-stage DN, based on parameters such as T1D duration, estimated glomerular filtration rate, microalbuminuria (MAU), urinary kidney injury molecule-1 (KIM-1), and episodes of diabetic ketoacidosis (DKA).

Results: Three clusters with distinct renal risk profiles were identified in children with T1D with or without DN. The risk group for DN development in T1D children showed moderate T1D duration, random MAU and DKA episodes, and slightly increased KIM-1 levels. The risk group for further DN progression in children with early DN was characterized by prolonged T1D duration, pronounced MAU, frequent DKA, hyperfiltration, and markedly elevated urinary KIM-1.

Conclusions: Cluster analysis allows early identification of the risk group for DN in children with T1D and the risk group for DN progression in children with early DN. Urinary KIM-1 may be implemented as a biomarker of tubular injury, providing opportunities for earlier intervention before clinical nephropathy develops.

KEY WORDS: diabetic nephropathy, kidney injury molecule-1, children

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INTRODUCTION

According to a study published in 2021, the global prevalence of diabetic nephropathy (DN) associated with type 1 diabetes mellitus (T1D) increased to 6,3 million cases in 2021. It is predicted that by 2041 this figure will reach 7 million cases, with incidence rates of 95,140 cases in 2021 and 115,000 expected by 2041 [1]. In Ukraine, there has been a significant increase in the age-standardized mortality rate from DN associated with T1D, from 1,9 per 100,000 population in 1990 to 12,4 in 2021, representing an increase of 560,2%. DN develops in 15-20% of children with T1D, and early manifestations may be asymptomatic [2–4].

In DN, functional disorders are associated with hyperglycemic damage to the glomerular endothelium, leading to increased basement membrane permeability and the onset of microalbuminuria (MAU). Activation of the renin-angiotensin-aldosterone system results in vasoconstriction, initial hyperfiltration, and a gradual decline in the estimated glomerular filtration rate (eGFR). Inflammatory processes are marked by elevated levels of the pro-inflammatory cytokines tumor necrosis factor (TNF-α) and interleukin-1 (IL-1), which induce fibrosis and interstitial degradation [3]. Oxidative stress causes injury to both the filtration and reabsorption epithelium of the nephron [4].

Various factors implicated in the development and progression of DN include metabolic and hemodynamic disorders, genetic predisposition, and inflammatory and fibrotic mechanisms [5]. However, glomerular lesions are considered the most dominant of the key pathomorphological changes in DN, determining the degree of impairment of the filtration capacity of the kidneys and closely correlating with the clinical picture. Modern studies also show that tubular-interstitial lesions are no less important in the pathogenesis of DN and may precede glomerular changes, especially in children. Several studies have highlighted the importance of kidney injury molecule-1 (KIM-1) as an early biomarker of tubular injury. Elevated urinary KIM-1 concentrations have been associated with the progression of chronic kidney disease and higher cardiovascular risk in patients with diabetes. However, most of the findings are based on studies in adults, and evidence in children with T1D and DN remains limited, emphasizing the importance of further research. In view of this, the search for new biomarkers and approaches to stratifying patients with T1D according to their risk of developing nephropathy is highly relevant [6,7].

AIM

To evaluate clinical and metabolic profiling parameters in children with T1D to identify markers associated with the early onset and progression of DN.

MATERIALS AND METHODS

To clarify the pathophysiological features and identify more informative diagnostic indicators, a cluster analysis was conducted in a cohort of 60 children (mean age [mean ± standard deviation] 13 ± 3 years; 17 [28,3%] boys and 43 [71,7%] girls), of whom 30 had T1D without signs of DN and 30 had early DN (pre-clinical, asymptomatic stage of kidney damage in T1D, with no major clinical symptoms yet apparent [8]). All patient data were documented, including medical history and systematic assessment. Urine albumin level (MAU) was assessed in 24-hour urine samples using standard methods (values less than 30 mg/24 h were considered normoalbuminuria, and 30–300 mg/24 h as MAU). The eGFR was calculated using the Schwartz formula [9]. The level of kidney injury molecule type 1 (KIM-1) in urine samples was determined using a commercial Human KIM-1 ELISA Kit (Cat. No. EH0210, 96 determinations; Wuhan Fine Biotech Co., Ltd, Wuhan, China).

The data were processed using Statistica v. 10.0 (originally developed by StatSoft Inc., USA), GraphPad Prism 10.5.0 for Windows (San Diego, CA, USA), and EZR v. 1.68. Quantitative data were expressed as median and interquartile range, and qualitative parameters as absolute and relative (%) frequency. Quantitative data were compared using the Mann-Whitney U test (for two unrelated samples) or the Kruskal-Wallis test with Dunn's post hoc test (for three unrelated samples). The frequency of binary parameters was compared using Fisher's exact test (for two unrelated samples) or Fisher's exact test with Bonferroni correction (for three unrelated samples).

Two-stage clustering was performed using Statistica v. 10.0 software. An intelligent clustering method was applied, in which the optimal number of clusters was determined automatically in two processes: initial clustering followed by hierarchical clustering. Hierarchical algorithms were used to estimate the optimal number of clusters based on silhouette width, log-likelihood distance calculation, and Bayesian Schwarz clustering. A p-value <0,05 was considered statistically significant (after applying Bonferroni correction).

ETHICS

This study was conducted in accordance with the principles outlined in the Declaration of Helsinki, as well as the recommendations established by ethical committee of the Bogomolets National Medical University (No. 4/2024). Prior to the start of the study, all participants received comprehensive information regarding the purpose, methods, potential risks, and benefits of participation. Participation in the study was based on the principle of informed consent, and all respondents signed an appropriate consent form before data collection began. Participants had the right to withdraw from the study at any time without providing a reason.

In the case of research involving minors or individuals with limited legal capacity, informed consent was obtained from their legal representatives.

In accordance with confidentiality standards, all data were collected anonymously and processed in compliance with the applicable data protection legislation, including the General Data Protection Regulation (GDPR), to ensure the protection of personal information. All information was used exclusively within the scope of this study and was aggregated for subsequent analysis of the results.

FRAMEWORK

The work is carried out within the framework of the initiative research work of the Bogomolets National Medical University (Kyiv, Ukraine) «Optimization of diagnostics and treatment and rehabilitation algorithms for the impact on the development of somatic diseases in children under martial law in Ukraine» (state registration number: 0125U000004; date: 2025-2027).

RESULTS

The analysis of clinical characteristics revealed that patients with DN were older and had a longer duration of diabetes compared to those with T1D without nephropathy (Table 1). The sex distribution was similar between groups. The frequency of diabetic ketoacidosis (DKA) and glycated hemoglobin (HbA1c) levels did not differ significantly. The T1D group was entirely represented by patients with normoalbuminuria, whereas in the DN group, 12 (40%) cases had normoalbuminuria. Moreover, the remaining 18 (60%) children with DN presented with MAU. The average urine albumin concentration (among detectable cases) was higher in the DN group. The eGFR was significantly increased in DN patients. Importantly, urine KIM-1 levels were almost two-fold higher in the DN group compared to the T1D group, indicating ongoing tubular injury in children with the pre-clinical stage of DN (Table 1).

In each group, three clusters were identified by remodeling cluster analysis based on five variables. The

Table 1. Clinical characteristics of patients in the studied groups

Characteristics	T1D (n=30)	DN (n=30)	p
Age, years	13 (9-15)	14 (13-16)	<0.05
Boys, n (%)	9 (30)	8 (27)	NS
Girls, n (%)	21 (70)	22 (73)	
DM duration, years	7 (4-9)	11 (8-12)	<0.001
DKA, episodes/year	2 (2-3)	3 (2-4)	NS
HbA1c, %	8.6 (6.5-10.6)	8.6 (7.8-9.5)	NS
Normoalbuminuria, n (%)	30 (100)	12 (40)	<0.0001
MAU, n (%)	0	18 (60)	
Urine albumin level, mg/24 h*	15.2 (14.5-17.1) N = 7**	83.0 (75.0-92.0) N = 18***	<0.0001
eGFR, ml/min/1,73 m^2	80.0 (75.0-85.0)	117.5 (114.0-120.0)	<0.001
Urine KIM-1, ng/ml	5.2 (2.8-6.2)	11.0 (8.4-13.2)	<0.001

Note: NS – non-significant; * – among the detectable cases; ** – urine albumin level (min-max) 6,1-20,1 mg/24 h; *** – urine albumin level (min-max) 65,0-95,0 mg/24 h

Source: compiled by the authors of this study

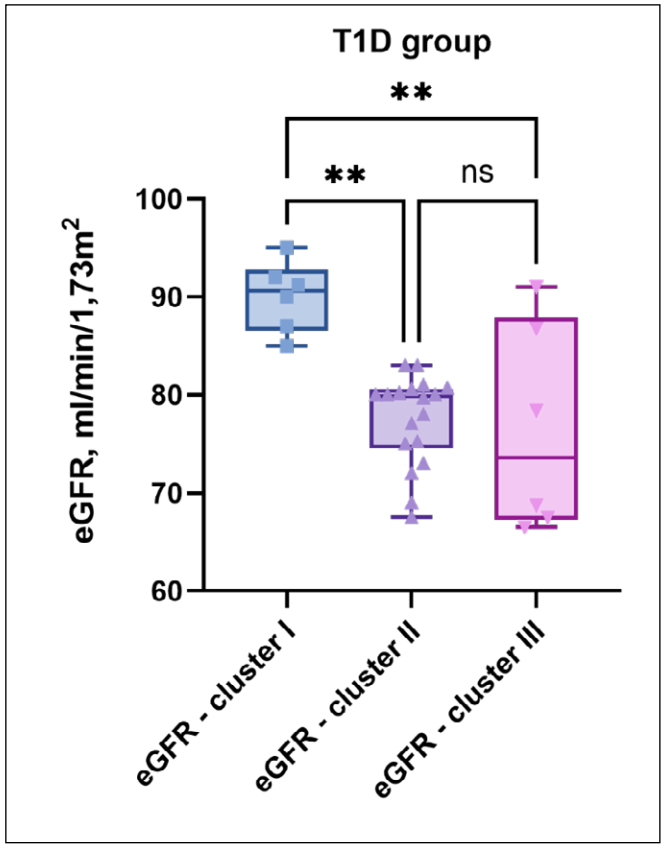

Fig. 1. eGFR levels in Clusters of the T1D group (box-and-whisker and dot plots [all the data]).
NS – non-significant; ** – p < 0,01
Picture taken by the authors

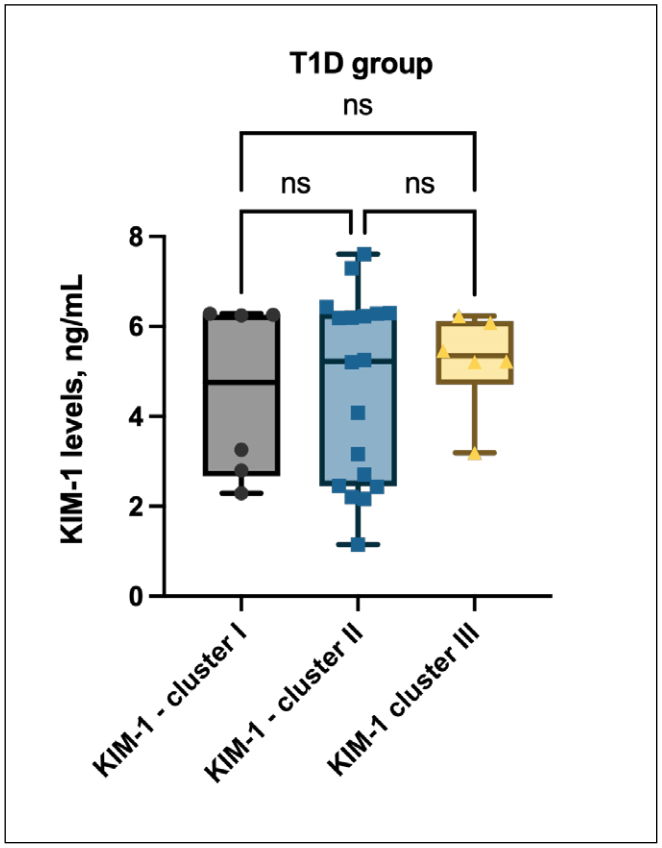

Fig. 2. Urine KIM-1 levels in the clusters of T1D group (box-and-whisker and dot plots [all the data])
NS – non-significant
Picture taken by the authors

clustered results were based on duration of diabetes, eGFR, MAU, DKA episodes per year, and KIM-1, a marker of tubular kidney injury. These five variables were selected because they reflect key clinical and pathophysiological dimensions of diabetic nephropathy progression. Duration of T1D is a major determinant of complication risk and disease stage. eGFR represents renal function status, and MAU is a well-established early marker of glomerular damage. The frequency of DKA episodes per year reflects the degree of metabolic instability

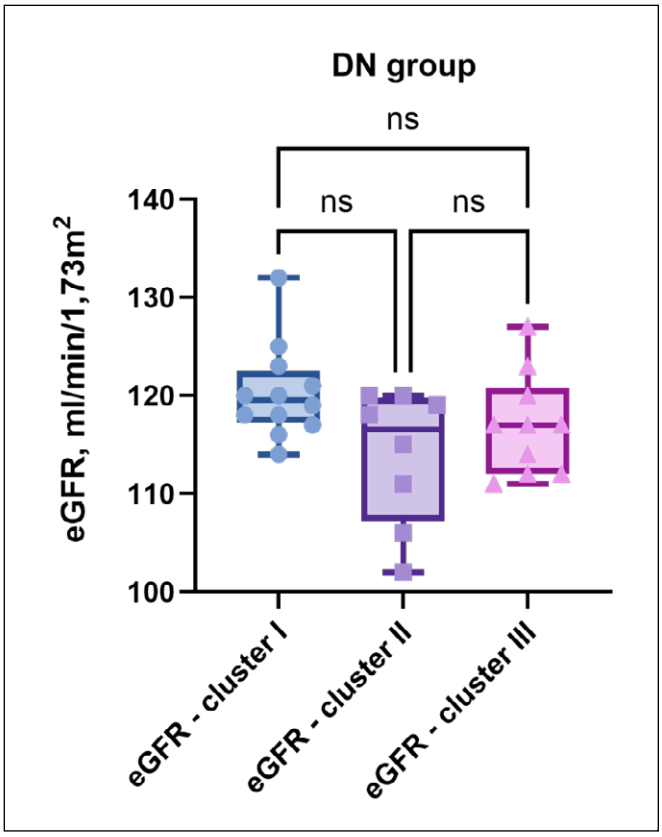

Fig. 3. eGFR levels in the Clusters of DN group (box-and-whisker and dot plots [all the data])
NS – non-significant
Picture taken by the authors

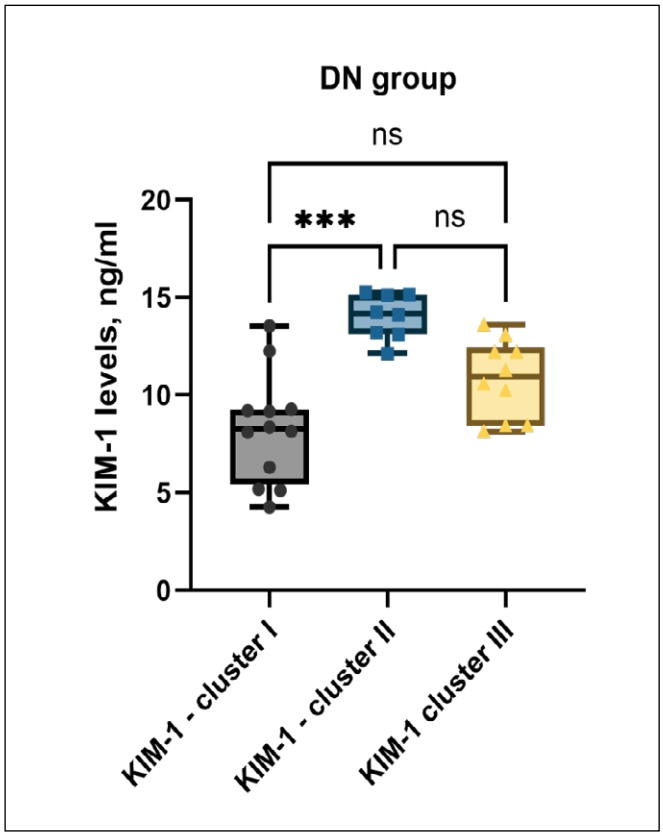

Fig. 4. Urine KIM-1 levels in clusters of DN group (box-and-whisker and dot plots [all the data])
NS – non-significant, *** – $p < 0,001$
Picture taken by the authors

and quality of T1D compensation, which contributes to vascular and renal injury. Finally, KIM-1 was included as a sensitive tubular injury biomarker, providing additional information on early kidney damage beyond conventional markers [10-12].

The analysis of T1D duration across clusters in the T1D group showed no statistically significant differences: the median disease duration was 6 (4–10) years in Cluster I (n = 6), 6 (2–9) years in Cluster II (n = 18), and 8 (7–8) years in Cluster III (n = 6) (p = 0,657).

The analysis of eGFR values across the three clusters in the T1D group revealed significant differences. Median eGFR was 90,6 (87,0–92,0) ml/min/1,73 m^2 in Cluster I, which was higher than the corresponding values in Cluster II (79,9 [75,0–80,6] ml/min/1,73 m^2; $p < 0,01$) and Cluster III (73,6 [67,5–86,8] ml/min/1,73 m^2; $p < 0,01$) (Fig. 1).

It should be noted that the majority of detectable cases of normoalbuminuria were observed in Cluster III (6 out of 7), despite only one case in Cluster I (p = 0,045) and the absence of such cases in Cluster II ($p < 0,0001$).

The frequency of DKA episodes per year did not differ significantly between clusters: Cluster I – 3 (1–3) episodes/year; Cluster II – 2 (2–3) episodes/year; and Cluster III – 3 (2–3) episodes/year (p = 0,567).

Finally, urine KIM-1 levels were comparable across the three clusters. Median values were 4,8 (2,8–6,3) ng/ml in Cluster I, 5,2 (2,5–6,3) ng/ml in Cluster II, and 5,3 (5,2–6,1) ng/ml in Cluster III (p = 0,973) (Fig. 2).

In the group of patients with DN, significant differences in the duration of type 1 diabetes were observed between the clusters: 8 (7–9) years in Cluster I (n=12), 13 (12–14) years in Cluster II (n=8), and 11 (9–11) years in Cluster III (n=10). The significant differences between Cluster I and Cluster II ($p < 0,001$), and between Cluster II and Cluster III ($p < 0,05$) were found.

In the DN group, the eGFR did not differ statistically significantly between the clusters: 119,5 (117,5–122,0) ml/min/1,73 m^2 in Cluster I, 116,5 (108,5–119,5) in Cluster II, and 117,0 (112,0–120,0) in Cluster III (p = 0,112) (Fig. 3).

The analysis of MAU cases between clusters in the DN group revealed certain differences. No cases of MAU were observed in Cluster I. At the same time, all children in Clusters II and III had MAU ($p < 0,0001$ vs. Cluster I for both comparisons). At the next step, higher urine albumin concentrations were observed in Cluster II compared to Cluster III: 92,0 (91,0–94,0) mg/24 h vs. 76,5 (71,0–80,0) mg/24 h, respectively ($p < 0,0001$).

The level of urine KIM-1 differed significantly between the clusters. Median values were 8,3 (5,8–9,2) ng/ml in Cluster I, 14,2 (13,2–15,1) ng/ml in Cluster II, and 11,0 (8,5–12,2) ng/ml in Cluster III. A significant difference was found between Clusters I and II ($p < 0,001$), while the difference between Clusters II and III showed a trend toward significance ($p = 0,052$) (Fig. 4).

The frequency of DKA episodes significantly differed between clusters of the DN group. In particular, median values were 1 (1–2) episodes/year in Cluster I, 4 (3–4) episodes/year in Cluster II, and 4 (3–4) episodes/year in Cluster III. The significant differences were revealed between Cluster I and Cluster II ($p < 0,01$), and between Cluster I and Cluster III ($p < 0,001$) (Fig. 5).

The distribution of clusters within the T1D and DN groups is presented in Fig. 6. In the T1D group, Cluster II predominated, comprising 60% of patients, while in the DN group, the largest proportion fell into Cluster I (40%). Clusters I and III in the T1D group were equal, each representing 20%. In the DN group, the proportions were 40% in Cluster I and 33% in Cluster III (Fig. 6).

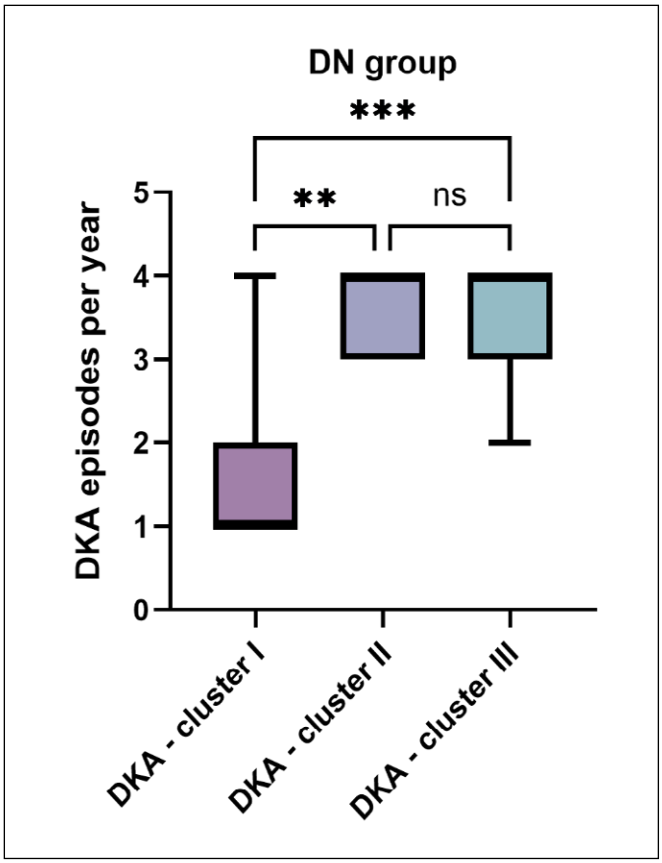

Fig. 5. DKA episodes/year in clusters of DN group (box-and-whisker plot)
NS – non-significant, ** – $p < 0,01$, *** – $p < 0,001$
Picture taken by the authors

DISCUSSION

This study revealed certain intercluster features regarding indicators of renal functional status and the course of T1D. The moderate average duration of T1D across all clusters of this group is consistent with other data [13]. In our study, Cluster III included patients with early signs of DN (decreased eGFR, albuminuria, and slightly elevated KIM-1).

Albuminuria increases with the progression of kidney disease and is therefore considered a marker of disease severity. MAU levels were significantly elevated in Cluster III, indicating initial impairment of glomerular barrier function in the patients of this cluster. Given the absence of clinical nephropathy in these patients, these changes can be interpreted as early predictors of kidney damage. Previous studies have shown that the first and highest peak of incidence occurs after 16 years of diabetes [14].

A modest increase in KIM-1 levels was observed in patients from all clusters. These findings are consistent with studies showing an association between urinary KIM-1 levels and the development of latent DN in children and adolescents with normoalbuminuria and T1D [15,16]. This also aligns with results from two pediatric cohorts with T1D of different durations, which found no significant differences in eGFR or MAU. These data support the idea that, in children without overt nephropathy, KIM-1 may serve as an independent marker of tubular damage without concurrent changes in glomerular function [17].

Cluster analysis allowed us to identify a group of children with T1D (Cluster III), in whom, despite a moderate duration of the disease, risk factors for the development of DN were already observed: episodes of microalbuminuria and DKA, decreased eGFR, and increased KIM-1. Similar to previous data [18], a decrease in eGFR may precede the appearance of MAU. This is consistent with findings showing the existence of preclinical forms of DN with an isolated increase in KIM-1 [18,19].

In our study, the highest T1D duration in the DN group was observed in Cluster II. The lowest average duration in Cluster I may indicate an early onset of nephropathy, even with a relatively shorter disease history, or a more intensive progression of kidney damage. The latter may be due to genetic, immunological, or metabolic factors, as also evidenced by other research groups [20].

The highest levels of KIM-1 were found in Cluster II, which directly indicates the predominance of tubulointerstitial kidney damage. The difference between Clusters II and III, along with the data from An et al., confirms the association of elevated KIM-1 with albuminuria, glycemic instability, and early glomerular changes. At the same time, regression of MAU is accompanied by a decrease in KIM-1 [21].

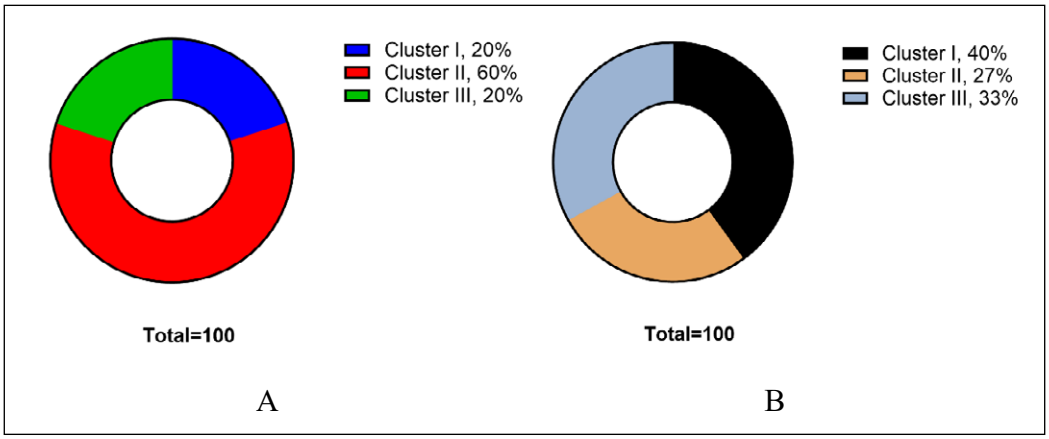

Fig. 6. The distribution (%) of clusters of patients in T1D (A) and DN (B) groups
Picture taken by the authors

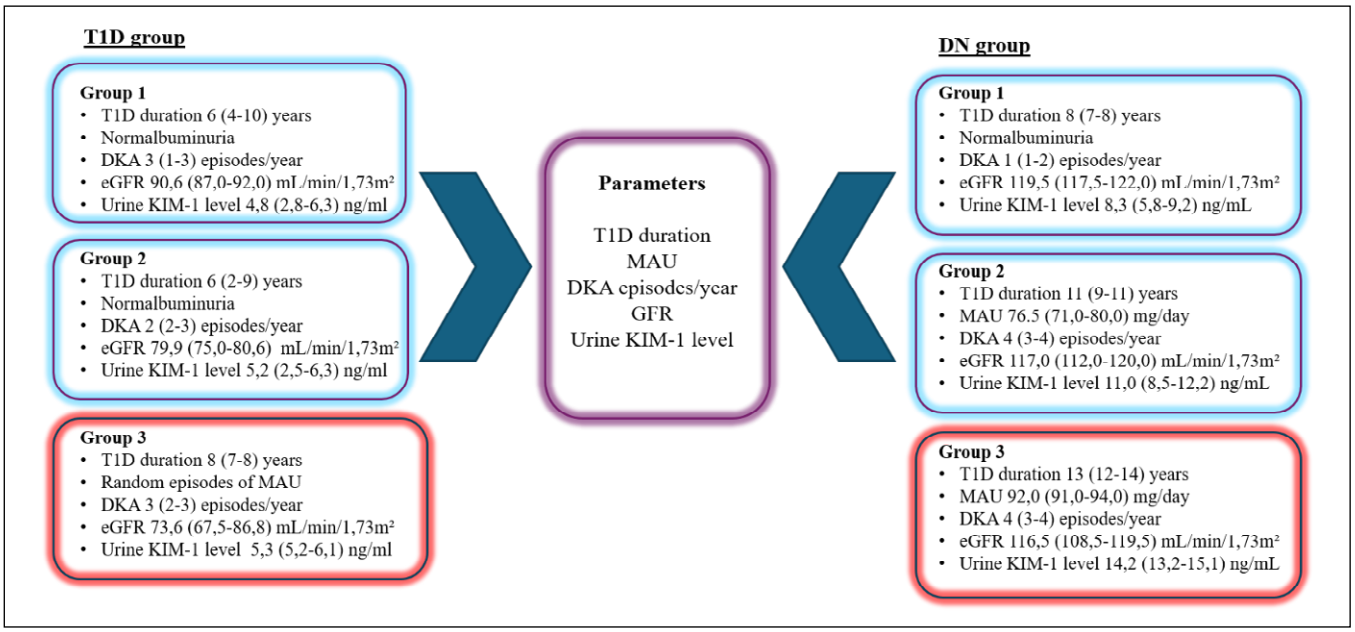

Fig. 7. Profiling of children with T1D according to the risk of developing DN based on clinical and biochemical parameters
Picture taken by the authors

MAU was not recorded in patients in Cluster I, suggesting that the glomeruli were still intact or the lesions had not yet manifested. In contrast, MAU was significantly increased in patients in Clusters II and III, indicating progressive damage to glomerular structures. The difference in MAU between Clusters II and III showed only a tendency toward significance, which may be related to the stage of disease or characteristics of treatment. Although MAU is a classic confirmatory test for the diagnosis of DN, not all patients develop macroalbuminuria.

The frequency of DKA episodes was statistically higher in Clusters II and III, potentially reflecting a more latent course of diabetes, insufficient glycemic control, and/or poor adherence to treatment, as also indicated by other studies [20–22].

The results of our study allowed us to identify two risk groups. One group, identified among T1D patients, was characterized by a moderate duration of T1D, sporadic MAU and DKA episodes, and KIM-1 levels exceeding the normal range. We speculate that this group can be considered a DKA risk group for further renal injury and DN development. In children from the DN group, we identified a risk group for subsequent DN progression, characterized by a longer duration of T1D, pronounced MAU, frequent DKA episodes, hyperfiltration, and markedly elevated urinary KIM-1 levels (Fig. 7).

Further studies should focus on a comprehensive analysis of the role of xanthine oxidase and KIM-1 as potential markers of early tubulointerstitial kidney damage in children with T1D and DN.

CONCLUSIONS

1. The cluster analysis verified distinct subgroups of children with T1D. Even with moderate disease duration, early renal changes were detected, including decreased eGFR, MAU, and elevated urinary KIM-1.

2. In children from the DN group, a risk group for subsequent DN progression, characterized by long T1D duration, pronounced MAU, frequent DKA episodes, hyperfiltration, and a marked rise in urinary KIM-1 levels.
3. Urinary KIM-1 measurement can considered as a complementary biomarker to MAU for early detection, monitoring, and risk stratification of DN in children with T1D.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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